Retroperitoneal hemorrhage after ureteroscopy without laser lithotripsy: an extreme example of an underreported event?

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Retroperitoneal hemorrhage and an associated hematoma are uncommon but potentially serious complications following ureteroscopy with laser lithotripsy. However,

Introduction

Ureteroscopy is a relatively safe diagnostic and therapeutic modality.¹ Recently, case studies and series have described the occurrence of renal subcapsular hematoma following uretersocopy and lithotripsy.²⁻⁶ In this article, we present a patient who developed a severe retroperitoneal hemorrhage and associated subcapsular hematoma following ureteroscopy without laser lithotripsy.

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Address correspondence to Dr. Jyoti D. Chouhan, 450 Clarkson Avenue, Box 79, Brooklyn, NY 11203 USA no reports of serious bleeding complications have been published regarding ureteroscopy without laser lithotripsy in the management of stone disease. We report of such a case here and then review the current literature in order to discuss the incidence, risk factors, and management of such events.

Key Words: ureteroscopy, hemorrhage, calculi

Case report

The following is a case report from an academic medical center. Patient consent for this report was obtained. In addition, the authors received approval from the medical center's Institutional Review Board.

An 82-year-old male with a history of hypertension and prostate cancer treated with brachytherapy in 2008 presented to the emergency department with right scrotal/groin pain, nausea, and chills for 5 days. The patient was afebrile and hemodynamically stable with a normal white blood cell count and an elevated serum creatinine (SCr) of 2.43 milligrams per deciliter (mg/dL) from a baseline of 1.5 mg/dL. Urinalysis was positive for leukocyte esterase, white blood cells and many bacteria. A computed tomography (CT) scan of the abdomen and pelvis revealed a 6 millimeter (mm) obstructing right mid-ureteral stone with associated mild hydronephrosis and proximal hydroureter, Figure 1. The patient was admitted, placed on empiric antibiotics, and underwent a cystoscopy with right ureteral stent placement with subsequent resolution of his symptoms. Of note, purulent material was seen draining from the ureteral orifice after stent placement. His urine culture was positive for Proteus mirabilis and he was discharged home with oral antibiotics based on sensitivities.

After discharge, urine culture the following month was positive for extended spectrum beta-lactamase (ESBL) producing Escherichia coli (E. coli). The patient was placed on oral antibiotics based on sensitivities with subsequent urine culture being negative. However, the procedure was delayed, and urine culture 2 months later again showed ESBL E. coli. This was treated with oral antibiotics and follow up urine culture was negative.

The patient then presented to ambulatory surgery for definitive treatment of his ureterolithiasis. Cystoscopy, removal of right ureter stent, right retrograde pyelogram and ureteroscopy with stone basket extraction were performed. Of note, the patient was not on any baseline anticoagulation. During the above procedure, a PTFE-nitinol guidewire with hydrophilic tip (Sensor wire, Boston Scientific,



Figure 1. Coronal image demonstrating the location of the patient's right ureteral stone.

Marlborough, MA, USA) was inserted into the right renal pelvis under fluoroscopic guidance and a 5 Fr open ended catheter was placed over the wire. After removal of the wire, a retrograde pyelogram was performed with no obvious filling defects noted. With replacement of the guidewire and removal of the catheter, the right ureteral orifice was dilated over the wire using an 8/10 dilator, and a second wire was passed up to the renal pelvis. The 8/10 dilator was removed, and a flexible ureteroscope was advanced over the second wire without difficulty. Once the ureteroscope was confirmed in the upper tract, the wire was subsequently removed. Irrigation during the procedure was provided with the use of a single action pump system. A distal ureteral stone was encountered, grasped and removed with a nitinol tipless basket (Zero-Tip Nitinol Stone Retrieval Basket, Boston Scientific, Marlborough, MA, USA). After removal of the ureteroscope, basket and stone, ureteropyeloscopy was performed with no further stone burden or signs of trauma found. A 6 Fr x 24 cm right ureteral stent was placed at the end of the procedure under direct and fluoroscopic guidance. There were no intraoperative complications or evidence of hemodynamic instability. The patient was extubated successfully and transferred to the post-anesthesia care unit in stable condition.

One hour postoperatively, the patient began complaining of abdominal pain and the urology service was notified. Additionally, nursing reported no urine output since arrival to the recovery room; vital signs revealed a heart rate between 140-150 beats per minute. A Foley catheter was placed without difficulty and ~100 mL of urine was drained. The patient subsequently reported improvement in his abdominal pain but vital signs showed new onset and progressive hypotension. A complete blood count (CBC) obtained at that time revealed a hemoglobin/hematocrit (H/H) of 7.3 grams per deciliter (g/dL)/22.1% from a preoperative baseline of 12.1 g/dL/35%. Repeat CBC 2 hours later confirmed the anemia with an H/H of 6.4 g/dL/19.3%. A central line was placed and the patient was resuscitated with intravenous crystalloids. As he was not responding to crystalloids appropriately, a continuous norepinephrine drip was initiated that evening. Overnight, two units of packed red blood cells (pRBCs) were given with a post-transfusion H/H of 8.1 g/dL/24.2%. During this time, blood cultures were drawn and the patient was placed on empiric piperacillin/tazobactam. An intensive care unit consult was placed and he was subsequently transferred to the intensive care unit.

The next morning, the patient was complaining of a dull, constant right flank pain but otherwise had no

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chest pain, shortness of breath, nausea or vomiting. His abdominal exam was soft, non-distended, and non-tender. Urine output had been 10-20 mL/hour after catheter placement despite a total of 5.2 liters of crystalloid and two units of pRBCs. He remained on a continuous norepinephrine drip with systolic blood pressure in the 90's to low 100's and heart rate stable at 120-130 beats per minute. Laboratory values were significant for acute kidney injury (SCr of 2.2 mg/dL from a baseline of 1.5 mg/dL), elevated liver enzymes (aspartate aminotransferase (AST) and alanine aminotransferase (ALT) of 535 international units per liter (IU/L) and 183 IU/L, respectively, and alkaline phosphatase of 132 IU/L) and a white blood cell count of 21.8 K/uL. He received an additional two units of pRBCs and underwent imaging with a non-contrast CT of the abdomen/pelvis that revealed interval development of a moderate sized right perinephric/ subcapsular hematoma. A hyperdense focus within the right renal parenchyma in the mid to upper pole was seen which likely represented hemorrhage, Figure 2. Additionally, there was a moderate to large amount of hemorrhage in the anterior pararenal space that extended into the pelvis and right inguinal hernia, Figure 3. The previously seen obstructing 6 mm right ureteral stone was no longer present. Later that day, his antibiotic regimen was changed to meropenem due to persistent leukocytosis.

On postoperative day 2, blood cultures showed gram-negative rods and the urine culture remained negative. His urine output improved to 40-150 mL/hr and his H/H remained stable at 10/28.9. The patient's



Figure 2. Axial image demonstrating the postoperative right renal hemorrhage.



Figure 3. Axial image demonstrating the postoperative extension of the retroperitoneal bleed into the right inguinal area (shown by the arrow).

tachycardia had improved to 100-110 beats per minute and the norepinephrine infusion was tapered and then discontinued in the evening. He was downgraded to the floor on postoperative day 4; blood cultures identified ESBL E. coli sensitive to meropenem. Laboratory studies showed an improving leukocytosis, a stable H/H (9.8 g/dL/29.3%) and normalized AST/ ALT (45 IU/L and 26 IU/L, respectively).

The patient was discharged home on postoperative day 8 with outpatient intravenous antibiotic infusion therapy to complete a 14 day course as per infectious disease recommendations. In urology clinic 2weeks later, he denied any flank pain, dysuria, fever, or chills. A follow up CT scan of the abdomen and pelvis without contrast was performed 6 weeks after discharge revealing complete resolution of the hemorrhage.

Discussion

Renal subcapsular hematoma and retroperitoneal hemorrhage have been well described following extracorporeal shock wave lithotripsy (ESWL), trauma, and a variety of other procedures. Renal subcapsular hematomas have only recently been acknowledged as complications following ureteroscopy with laser lithotripsy. Bansal et al first described a case of renal subcapsular hematoma following ureteroscopy with pneumatic lithotripsy for a right lower ureteric calculus with moderate dilation of the pelvicalyceal system.² The patient presented with severe right flank pain and tenderness 12 hours postoperatively and required percutaneous drainage of the hematoma. Xu et al reported a case of renal subcapsular hematoma following laser lithotripsy of a left kidney stone with no evidence of hydronephrosis.³ This patient required urgent angiography and embolization after presenting 1 hour postop with tachycardia and a decreasing hematocrit refractory to multiple transfusions.

Several recent case series have better described the incidence and risk factors of renal subcapsular hematoma. Bai et al reviewed 2848 patients treated with Holmium - Yyttrium-aluminum-garnet (Ho-YAG) laser lithotripsy from 2003-2010 and found 11 patients with renal subcapsular hematoma (0.39%).4 All patients presented with flank pain or a palpable mass accompanied by hematuria, fever, diffuse abdominal pain, or shock. The average time to presentation of postoperative symptoms was 6 hours. Three patients were treated conservatively. Six patients improved with drainage, and two patients underwent open surgery. All hematomas had resolved at 6 months follow up. Their analysis showed that risk factors for developing renal subcapsular hematoma included larger stones, more severe ipsilateral hydronephrosis, longer operation duration, and higher perfusion pressure of hydraulic irrigation.

Chiu et al looked at 1114 cases of Ho-YAG laser lithotripsies from 2007 and 2012 and found four patients with renal subcapsular hematoma (0.36%).⁵ All four patients had proximal upper ureteral stones and three of the four had thin renal cortices. All presented with fever, flank pain, and a drop in hemoglobin. Three patients presented within 2 days and one presented on postop day 20. Meng et al found eight cases of renal subcapsular hematoma out of 1918 (0.42%) total Ho-YAG laser lithotripsies.⁶ All eight had moderate to severe hydronephrosis and all eight presented with flank pain from 0-18 hours postop. Analysis showed a significance in the patient's history of prior ESWL, renal operation, and hypertension between those with and without renal subcapsular hematoma.

Most recently, Kozminski et al reviewed 877 renal units exposed to Ho-YAG laser for lithotripsy and found four cases of renal subcapsular hematoma and three cases of perinephric hematoma.⁷ They found a significant correlation of hematoma with the female gender, preoperative hypertension, preoperative ureteral stenting, intraoperative ureteral sheath use, and postoperative ureteral stenting.

There are several theories for the development of renal subcapsular hematomas after ureteroscopy. Guidewire trauma to the pelvicalyceal system and renal parenchyma is one proposed mechanism. Bansal et al described the possibility of raised intrarenal pressure leading to forniceal rupture and separation of the capsule from the parenchyma.² Bai et al confirmed this as one of their two patients who required open surgery was found to have ruptured fornices.⁴ Their findings of severe hydronephrosis and increased perfusion pressure as risk factors for renal subcapsular hematoma are consistent with this theory as well. Severe hydronephrosis causes kinking and stretching of the nearby vessels, which can suddenly expand and rupture upon recanalization of the ureter. A patient's medical history and renal disease history may be a factor as well.⁶⁷ Urinary tract infection might have an important role due to inflammatory damage.³

Our case shares several characteristics with the aforementioned studies. Our patient initially presented with a ureteral calculus and associated mild hydronephrosis. The hydronephrosis likely resolved in the subsequent 5 month timeframe before ureteroscopic intervention due to initial stent placement but there was no imaging available to demonstrate that. It's possible that a singe occurrence of hydronephrosis can have long term effects on the renal unit. The ureteroscopy was also delayed due to a UTI, which may have further weakened the parenchyma and surrounding vessels. However, another month had passed between the UTI and the ureteroscopy.

The incidence of symptomatic hematoma in the four case series was similar (0.36%-0.8%). These numbers may underestimate the true incidence as smaller hematomas are asymptomatic and resolve spontaneously without any work up. Nevertheless, renal subcapsular hematoma and PH can be lifethreatening and physicians need to have a high index of suspicion. Almost every patient reviewed presented with flank pain with or without fever, signs of shock, a palpable mass, and a decreasing hematocrit. The time to presentation was usually less than 24-48 hours although a few patients presented upwards of a month later. Our patient presented much more acutely. Just 1 hour after the operation, he was tachycardic with a right flank pain. His sudden onset of symptoms and the large drop in hematocrit represented a hemorrhage into a much larger potential space.

Importantly, this case demonstrates that ureteroscopy without lithotripsy can result in hematoma and hemorrhage. While the transfer of energy of different lithotripsic modalities may play some role in ureter and renal parenchymal damage, the complications in our patient could be attributed to the high perfusion pressure. We did not use an access sheath during the procedure, which has been found to reduce the intrarenal pelvic pressure.⁸ Although Kozminski et al demonstrated an association between sheath use and hematoma formation, they discussed the possibility that access sheaths were reserved Retroperitoneal hemorrhage after ureteroscopy without laser lithotripsy: an extreme example of an underreported event?

for harder stones and greater stone burdens which would have required more laser utilization and stone manipulation.⁷

The treatment of renal subcapsular hematoma and PH should be dependent on the patient's hemodynamic status, renal function, and presence of infection. Conservative management should be attempted with fluids, antibiotics, and transfusions as needed with frequent monitoring of vital signs, creatinine, and hemoglobin/hematocrit. A contained hematoma in the subcapsular space may impair kidney function and require percutaneous drainage. Drainage may also be necessary for persistent and progressive symptoms. Embolization and open surgery can be reserved for cases with active and uncontrolled hemorrhage refractory to the above measures.

Unlike renal subcapsular hematoma, retroperitoneal hemorrhage has not been a well-described complication of ureteroscopy. Watterson et al examined the safety of Ho-YAG lithotripsy in patients with bleeding diatheses.⁹ Of the 30 patients studied, two received concomitant treatment with electrohydraulic lithotripsy. One of those two developed a retroperitoneal hemorrhage requiring transfusion. They believed that the acoustic shock waves were transmitted deep to the urothelium as no hematuria was noted postoperatively.

In our patient, the retroperitoneal hemorrhage likely represents spread from the subcapsular hematoma. Management is similar to that of isolated renal subcapsular hematoma, although more attention must be paid to the larger potential space for hemorrhage. Our patient was treated conservatively with vasopressors, fluids, transfusions, and antibiotics. His initial hemodynamic instability, likely exacerbated by septic shock, stabilized and no acute interventions were needed. Synchronous damage to another artery was unlikely because no lithotripsy was used and the clinical picture improved with conservative treatment. This case was assigned a Grade IVb Clavien-Dindo classification for multi-organ dysfunction, highlighting the possible severity of these complications.

In conclusion, renal subcapsular hematomas are uncommon but potentially serious complications of ureteroscopy. Further studies can better identify techniques to reduce their risk. Retroperitoneal hemorrhage from ureteroscopy is more rare and may be secondary to damage of other vessels or spread from a large renal subcapsular hematoma. Treatment of both depends on an assessment of hemodynamic status, kidney function, and response to conservative management. We suggest that patients should be made aware of these risks before any type of ureteroscopic procedure.

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